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NEWS 1		Web Page URLs for STN Seminar Schedule - N. America
NEWS 2	Jan 25	BLAST(R) searching in REGISTRY available in STN on the Web
NEWS 3	Jan 25	Searching with the P indicator for Preparations
NEWS 4	Jan 29	FSTA has been reloaded and moves to weekly updates
NEWS 5	Feb 01	DKILIT now produced by FIZ Karlsruhe and has a new update frequency
NEWS 6	Feb 19	Access via Tymnet and SprintNet Eliminated Effective 3/31/02
NEWS 7	Mar 08	Gene Names now available in BIOSIS
NEWS 8	Mar 22	TOXLIT no longer available
NEWS 9	Mar 22	TRCTHERMO no longer available
NEWS 10	Mar 28	US Provisional Priorities searched with P in CA/CAplus and USPATFULL
NEWS 11	Mar 28	LIPINSKI/CALC added for property searching in REGISTRY
NEWS 12	Apr 02	PAPERCHEM no longer available on STN. Use PAPERCHEM2 instead.
NEWS 13	Apr 08	"Ask CAS" for self-help around the clock
NEWS 14	Apr 09	BEILSTEIN: Reload and Implementation of a New Subject Area
NEWS 15	Apr 09	ZDB will be removed from STN

NEWS EXPRESS	February 1 CURRENT WINDOWS VERSION IS V6.0d, CURRENT MACINTOSH VERSION IS V6.0a(ENG) AND V6.0Ja(JP), AND CURRENT DISCOVER FILE IS DATED 05 FEBRUARY 2002
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NEWS INTER	General Internet Information
NEWS LOGIN	Welcome Banner and News Items
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FILE 'HOME' ENTERED AT 09:50:46 ON 17 APR 2002

=> file medline biosis embase caplus uspatfull

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                                                           ENTRY    SESSION
FULL ESTIMATED COST                                         0.21       0.21

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=> s cyp24 (p) (nuclear (a) receptor) (p) (reporter (s) gene)

L1             4 CYP24 (P) (NUCLEAR (A) RECEPTOR) (P) (REPORTER (S) GENE)

=> dup rem l1

PROCESSING COMPLETED FOR L1
L2             1 DUP REM L1 (3 DUPLICATES REMOVED)

=> d l2 total ibib kwic


L2   ANSWER 1 OF 1     MEDLINE                              DUPLICATE 1
ACCESSION NUMBER:    2000259547     MEDLINE
DOCUMENT NUMBER:     20259547   PubMed ID: 10797570
TITLE:               Natural metabolites of 1alpha,25-dihydroxyvitamin D(3)
                     retain biologic activity mediated through the vitamin D
                     receptor.
AUTHOR:              Harant H; Spinner D; Reddy G S; Lindley I J
CORPORATE SOURCE:    Department of Inflammatory Diseases, Novartis Research
                     Institute, Vienna, Austria..
Hanna.Harant@pharma.novartis.c
                     om
SOURCE:              JOURNAL OF CELLULAR BIOCHEMISTRY, (2000 Apr) 78 (1)
112-20.
                     Journal code: HNF; 8205768. ISSN: 0730-2312.
PUB. COUNTRY:        United States
                     Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:            English
FILE SEGMENT:        Priority Journals
ENTRY MONTH:         200007
ENTRY DATE:          Entered STN: 20000720
                     Last Updated on STN: 20000720
                     Entered Medline: 20000710
AB   . . . mediates many of its effects through the intranuclear vitamin D
     receptor (VDR, NR1I1), that belongs to the large superfamily of
     nuclear receptors. Vitamin D receptor can directly
     regulate gene expression by binding to vitamin D response
     elements (VDREs) located in promoter or enhancer regions of various
     genes. Although numerous synthetic analogs of 1alpha,25(OH)(2)D(3)
     have been analysed for VDR binding and transactivation of VDRE-driven
     gene expression, the biologic activity of many naturally occurring
     metabolites has not yet been analyzed in detail. We therefore studied
the.
     . . (1alpha(OH)-24,25,26,27-tetranor-23-COOH-D(3); calcitroic acid)
```

using the human G-361 melanoma cell line. Cells were cotransfected with a VDR expression plasmid and luciferase **reporter gene** constructs driven by two copies of the VDRE of either the mouse osteopontin promoter or the 1alpha,25(OH)(2)D(3) 24-hydroxylase (**CYP24**) promoter. Treatment with 1alpha,25(OH)(2)D(3) or the metabolites 1alpha,24R,25(OH)(3)D(3), 1alpha,25(OH)(2)-3-epi-D(3), and 1alpha,23S,25(OH)(3)D(3) resulted in transactivation of both constructs in a time-. . . effect was observed even for calcitroic acid in the presence of overexpressed VDR. The metabolites that were active in the **reporter gene** assay also induced expression of **CYP24** mRNA in the human keratinocyte cell line HaCaT, although with less potency than the parent hormone. A ligand-binding assay based on nuclear extracts from COS-1 cells overexpressing human VDR demonstrated that the metabolites, although active in the **reporter gene** assay, were much less effective in displacing [(3)H]-labeled 1alpha,25(OH)(2)D(3) from VDR than the parent hormone. Thus, we report that several. . .

=> s 24-OHase (p) (nuclear (a) receptor) (p) (reporter (s) gene)

L3 0 24-OHASE (P) (NUCLEAR (A) RECEPTOR) (P) (REPORTER (S) GENE)

=> s 24-hydroxylase (p) (nuclear (a) receptor) (p) (reporter (s) gene)

L4 8 24-HYDROXYLASE (P) (NUCLEAR (A) RECEPTOR) (P) (REPORTER (S) GENE)

=> dup rem l4

PROCESSING COMPLETED FOR L4
L5 2 DUP REM L4 (6 DUPLICATES REMOVED)

=> d l5 total ibib kwic

L5 ANSWER 1 OF 2 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2000259547 MEDLINE
DOCUMENT NUMBER: 20259547 PubMed ID: 10797570
TITLE: Natural metabolites of 1alpha,25-dihydroxyvitamin D(3) retain biologic activity mediated through the vitamin D receptor.
AUTHOR: Harant H; Spinner D; Reddy G S; Lindley I J
CORPORATE SOURCE: Department of Inflammatory Diseases, Novartis Research Institute, Vienna, Austria..
Hanna.Harant@pharma.novartis.com
SOURCE: JOURNAL OF CELLULAR BIOCHEMISTRY, (2000 Apr) 78 (1) 112-20.
Journal code: HNF; 8205768. ISSN: 0730-2312.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200007
ENTRY DATE: Entered STN: 20000720
Last Updated on STN: 20000720
Entered Medline: 20000710

AB . . . mediates many of its effects through the intranuclear vitamin D receptor (VDR, NR1I1), that belongs to the large superfamily of **nuclear receptors**. Vitamin D receptor can directly regulate **gene** expression by binding to vitamin D response elements (VDREs) located in promoter or enhancer regions of various **genes**. Although numerous synthetic analogs of 1alpha,25(OH)(2)D(3)

have been analysed for VDR binding and transactivation of VDRE-driven **gene** expression, the biologic activity of many naturally occurring metabolites has not yet been analyzed in detail. We therefore studied the.

. . (1alpha(OH)-24,25,26,27-tetranor-23-COOH-D(3); calcitroic acid) using the human G-361 melanoma cell line. Cells were cotransfected with a VDR expression plasmid and luciferase **reporter gene** constructs driven by two copies of the VDRE of either the mouse osteopontin promoter or the 1alpha,25(OH)(2)D(3) **24-hydroxylase** (CYP24) promoter. Treatment with 1alpha,25(OH)(2)D(3) or the metabolites 1alpha,24R,25(OH)(3)D(3), 1alpha,25(OH)(2)-3-epi-D(3), and 1alpha,23S,25(OH)(3)D(3) resulted in transactivation of both constructs in a. . . effect was observed even for calcitroic acid in the presence of overexpressed VDR. The metabolites that were active in the

reporter gene assay also induced expression of CYP24 mRNA in the human keratinocyte cell line HaCaT, although with less potency

than the. . . ligand-binding assay based on nuclear extracts from COS-1

cells overexpressing human VDR demonstrated that the metabolites, although

active in the **reporter gene** assay, were much less effective in displacing [(3)H]-labeled 1alpha,25(OH)(2)D(3) from VDR than the parent hormone. Thus, we report that several. . .

L5 ANSWER 2 OF 2 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 1999278410 MEDLINE
DOCUMENT NUMBER: 99278410 PubMed ID: 10347199
TITLE: Antagonistic action of novel 1alpha,25-dihydroxyvitamin D3-26, 23-lactone analogs on differentiation of human leukemia cells (HL-60) induced by 1alpha,25-dihydroxyvitamin D3.
AUTHOR: Miura D; Manabe K; Ozono K; Saito M; Gao Q; Norman A W; Ishizuka S
CORPORATE SOURCE: Safety Research Department, Teijin Institute for Bio-Medical Research, 4-3-2 Asahigaoka, Hino, Tokyo 191-8512, Japan.
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1999 Jun 4) 274 (23) 16392-9.
Journal code: HIV; 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199907
ENTRY DATE: Entered STN: 19990714
Last Updated on STN: 19990714
Entered Medline: 19990701

AB . . . novel 1alpha,25-dihydroxyvitamin D3-26,23-lactone (1alpha,25-lactone) analogues on human promyelocytic leukemia cell (HL-60)

differentiation using the evaluation system of the vitamin D **nuclear receptor** (VDR)/vitamin D-responsive element (DRE)-mediated genomic action stimulated by 1alpha,25-dihydroxyvitamin D3 (1alpha,25(OH)2D3) and its analogues. We found that the 1alpha,25-lactone analogues. . . effective antagonist of both 1alpha,25(OH)2D3 (10(-8) M)

mediated induction of p21(WAF1, CIP1) in HL-60 cells and activation of the

luciferase **reporter** assay in COS-7 cells transfected with cDNA containing the DRE of the rat 25(OH)D3-**24-hydroxylase** **gene** and cDNA of the human VDR. Collectively the results strongly suggest that our novel 1alpha,25-lactone analogues, TEI-9647 and TEI-9648,

are. . .

=> log y

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	36.85	37.06

STN INTERNATIONAL LOGOFF AT 09:57:11 ON 17 APR 2002

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NEWS	7	Mar 08	Gene Names now available in BIOSIS
NEWS	8	Mar 22	TOXLIT no longer available
NEWS	9	Mar 22	TRCTHERMO no longer available
NEWS	10	Mar 28	US Provisional Priorities searched with P in CA/CAplus and USPATFULL
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NEWS	12	Apr 02	PAPERCHEM no longer available on STN. Use PAPERCHEM2 instead.
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FILE 'HOME' ENTERED AT 10:54:08 ON 17 APR 2002

=> file medline biosis embase caplus uspatfull

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COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
                                                           ENTRY    SESSION
FULL ESTIMATED COST                                         0.21       0.21

FILE 'MEDLINE' ENTERED AT 10:54:20 ON 17 APR 2002

FILE 'BIOSIS' ENTERED AT 10:54:20 ON 17 APR 2002
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=> s (expression (a) cloning) (p) (nuclear (a) hormone (a) receptor) (p)
(reporter (a) gene)

   4 FILES SEARCHED...
L1            0 (EXPRESSION (A) CLONING) (P) (NUCLEAR (A) HORMONE (A)
RECEPTOR)
                (P) (REPORTER (A) GENE)

=> s (expression (a) clon?) (p) (nuclear (a) receptor) (p) (reporter (a) gene)

   3 FILES SEARCHED...
L2            0 (EXPRESSION (A) CLON?) (P) (NUCLEAR (A) RECEPTOR) (P)
(REPORTER
                (A) GENE)

=> s expression (p) clon? (p) (nuclear (a) receptor) (p) (reporter (a) gene)

   3 FILES SEARCHED...
L3           88 EXPRESSION (P) CLON? (P) (NUCLEAR (A) RECEPTOR) (P) (REPORTER
                (A) GENE)

=> dup rem l3

PROCESSING COMPLETED FOR L3
L4            33 DUP REM L3 (55 DUPLICATES REMOVED)

=> d l4 total ibib kwic


L4   ANSWER 1 OF 33  USPATFULL
ACCESSION NUMBER:         2002:43573  USPATFULL
TITLE:                    Methods and compositions for sensitive and rapid,
                          functional identification of genomic polynucleotides
                          and use for cellular assays in drug discovery
INVENTOR(S):              Whitney, Michael A., La Jolla, CA, UNITED STATES
                          Xanthopoulos, Kleanthis, La Jolla, CA, UNITED STATES
                          Nelson, David, San Diego, CA, UNITED STATES
                          Negulescu, Paul, Solana Beach, CA, UNITED STATES
                          Craig, Frank, Glasgow, UNITED KINGDOM
                          Foulkes, J. Gordon, Encinitas, CA, UNITED STATES
PATENT ASSIGNEE(S):       Aurora Biosciences Corporation (U.S. corporation)

                               NUMBER         KIND    DATE
                          ---------------- ------ --------
PATENT INFORMATION:       US 2002025940      A1   20020228
```

APPLICATION INFO.: US 2001-772114 A1 20010126 (9)
RELATED APPLN. INFO.: Continuation of Ser. No. US 1998-47862, filed on 25 Mar
1998, PENDING Continuation-in-part of Ser. No. US 1998-21974, filed on 11 Feb 1998, ABANDONED A 371 of International Ser. No. WO 1997-US17395, filed on 26 Sep
1997, UNKNOWN
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Lisa A. Haile, Ph.D., Gray Cary Ware & Freidenrich LLP,
4365 Executive Drive, Suite 1600, San Diego, CA, 92121-2189
NUMBER OF CLAIMS: 143
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 4 Drawing Page(s)
LINE COUNT: 4442
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
DETD . . . cells can be separated by FACS. These two cell populations can be treated with potential modulators and changes in gene **expression** can be monitored using ratio-metric fluorescent readout. Pools of **clones** will be isolated that show either up- or down-regulation of **reporter gene** **expression**. Target genes from responding **clones** can then be identified. In addition, by being able to separate expressing and non-expressing cells at different time points after. . . Specifically, it will provide a means to identify downstream genes which are transcriptionally regulated by a variety of molecules including, **nuclear receptors**, cytokine receptors or transcription factors.

L4 ANSWER 2 OF 33 USPATFULL
ACCESSION NUMBER: 2000:150147 USPATFULL
TITLE: Specific expression vectors and methods of use
INVENTOR(S): Roop, Dennis R., Houston, TX, United States
Rothnagel, Joseph A., Houston, TX, United States
Greenhalgh, David A., Houston, TX, United States
PATENT ASSIGNEE(S): Baylor College of Medicine, Houston, TX, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6143727		20001107
APPLICATION INFO.:	US 1995-458240		19950605 (8)

RELATED APPLN. INFO.: Division of Ser. No. US 1993-146930, filed on 1 Nov 1993, now patented, Pat. No. US 5958764 which is a continuation-in-part of Ser. No. US 1993-145388, filed on 29 Oct 1993, now abandoned which is a continuation-in-part of Ser. No. US 1992-876286, filed on 30 Apr 1992, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Hauda, Karen M.
LEGAL REPRESENTATIVE: Lyon & Lyon LLP
NUMBER OF CLAIMS: 13
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 11 Drawing Figure(s); 11 Drawing Page(s)
LINE COUNT: 2126
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
SUMM The 5' regulatory regions of four human epidermal keratin genes, K5, K6, K10 and K14, have been **cloned** into vectors to drive **expression** of the CAT **reporter gene**. These constructs were transfected into epithelial cells along with vectors

expressing **nuclear receptors** for retinoic acid and thyroid hormone. (Tomic, et al., Cell Reg., Vol. 1, pp. 965-973 (1990)).

This study demonstrated that. . . the human K1 gene responded to elevated levels of calcium in order to induce both mouse K1 and human K1

expression.

L4 ANSWER 3 OF 33 USPATFULL

ACCESSION NUMBER: 2000:121281 USPATFULL
TITLE: Methods to screen for transcription factor-coactivator interactions
INVENTOR(S): Kushner, Peter J., San Francisco, CA, United States
Webb, Paul, San Francisco, CA, United States
Uht, Rosalie M., San Francisco, CA, United States
PATENT ASSIGNEE(S): The Regents of the University of California, Oakland, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6117638		20000912
APPLICATION INFO.:	US 1998-54238		19980402 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 1997-43059P	19970404 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: McKelvey, Terry
LEGAL REPRESENTATIVE: Skjerven, Morrill, MacPherson, Franklin & Friel, LLP, Hunter, Esq., Tom
NUMBER OF CLAIMS: 14
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 15 Drawing Figure(s); 10 Drawing Page(s)
LINE COUNT: 1364
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD The invention also provides methods for identifying previously unknown coactivators that are involved in **nuclear receptor** -mediated transcriptional regulation. An **expression** library of cDNA molecules is prepared from mRNA obtained from a cell in which a gene of interest is expressed. **Expression** screening is described in, for example, Ausubel, supra. The **expression** vector used for the library includes a DNA binding domain coding region adjacent to the insertion site for the cDNA **clones**. The **expression** library DNAs are co-introduced into a host cell with a transcription factor polypeptide, which can also be provided by means of **expression** of a heterologous gene. A hormone or analog that binds to the transcription factor polypeptide is also introduced into the cells, thus activating the transcription factor polypeptide. In a preferred embodiment, the host cells also contain a **reporter gene** that is operably linked to a response element that corresponds to the DNA binding domain encoded by the **expression** vector. **Clones** that encode an activation domain of a coactivator will trigger **expression** of genes that are operably linked to the response element.

L4 ANSWER 4 OF 33 USPATFULL

ACCESSION NUMBER: 2000:54081 USPATFULL
TITLE: Keratin K1 expression vectors and methods of use
INVENTOR(S): Roop, Dennis R., Houston, TX, United States
Rothnagel, Joseph A., Houston, TX, United States
Greenhalgh, David A., Houston, TX, United States
Yuspa, Stuart H., Bethesda, MD, United States
PATENT ASSIGNEE(S): Baylor College of Medicine, Houston, TX, United States (U.S. corporation)
The United States of America as represented by the

Department of Health and Human Services, Washington, DC, United States (U.S. government)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6057298		20000502
APPLICATION INFO.:	US 1995-452872		19950530 (8)

RELATED APPLN. INFO.: Division of Ser. No. US 1993-147777, filed on 1 Nov 1993, now patented, Pat. No. US 5914265 which is a continuation-in-part of Ser. No. US 1993-145387, filed on 29 Oct 1993, now abandoned which is a continuation-in-part of Ser. No. US 1992-876289, filed on 30 Apr 1992, now abandoned

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Hauda, Karen M.
LEGAL REPRESENTATIVE: Lyon & Lyon LLP
NUMBER OF CLAIMS: 23
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 10 Drawing Figure(s); 7 Drawing Page(s)
LINE COUNT: 3628
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM The 5' regulatory regions of four human epidermal keratin genes, K5, K6, K10 and K14, have been **cloned** into vectors to drive **expression** of the CAT **reporter gene**. These constructs were transfected into epithelial cells along with vectors expressing **nuclear receptors** for retinoic acid and thyroid hormone. (Tomic, et al., Cell Reg., Vol. 1, pp. 965-973 (1990)).

This study demonstrated that. . . the human K1 gene responded to elevated levels of calcium in order to induce both mouse K1 and human K1 **expression**.

L4 ANSWER 5 OF 33 MEDLINE DUPLICATE 1

ACCESSION NUMBER: 2001022555 MEDLINE
DOCUMENT NUMBER: 20461445 PubMed ID: 11005856
TITLE: CXR, a chicken xenobiotic-sensing orphan nuclear receptor, is related to both mammalian pregnane X receptor (PXR) and constitutive androstane receptor (CAR).
AUTHOR: Handschin C; Podvinec M; Meyer U A
CORPORATE SOURCE: Division of Pharmacology/Neurobiology, Biozentrum of the University of Basel, Klingelbergstrasse 70, CH-4056 Basel, Switzerland.
SOURCE: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (2000 Sep 26) 97 (20) 10769-74.

Journal code: PV3. ISSN: 0027-8424.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-AF276753
ENTRY MONTH: 200011
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20001103

AB **Nuclear receptors** constitute a large family of ligand-modulated transcription factors that mediate cellular responses to small lipophilic molecules, including steroids, retinoids, fatty acids, and exogenous ligands. Orphan **nuclear receptors** with no known endogenous ligands have been discovered to regulate drug-mediated induction of cytochromes P450 (CYP), the major drug-metabolizing enzymes. Here, we report the **cloning** of an orphan **nuclear**

receptor from chicken, termed chicken xenobiotic receptor (CXR), that is closely related to two mammalian xenobiotic-activated receptors, the pregnane X receptor (PXR) and the constitutive androstane receptor (CAR). **Expression** of CXR is restricted to tissues where drug induction of CYPs predominantly occurs, namely liver, kidney, small intestine, and colon.. . . A variety of drugs, steroids, and chemicals activate CXR in CV-1 monkey cell transactivation assays. The same agents induce PBRU-dependent **reporter gene expression** and CYP2H1 transcription in a chicken hepatoma cell line. These results provide convincing evidence for a major role of CXR in the regulation of CYP2H1 and add a member to the family of xenobiotic-activated orphan **nuclear receptors**.

L4 ANSWER 6 OF 33 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 2000485433 MEDLINE
DOCUMENT NUMBER: 20486906 PubMed ID: 11034093
TITLE: Down-Regulation of prostate-specific antigen expression by ligands for peroxisome proliferator-activated receptor gamma in human prostate cancer.
AUTHOR: Hisatake J I; Ikezoe T; Carey M; Holden S; Tomoyasu S; Koeffler H P
CORPORATE SOURCE: Division of Hematology/Oncology Cedars-Sinai Medical Center, University of California-Los Angeles School of Medicine, 90048, USA.
SOURCE: CANCER RESEARCH, (2000 Oct 1) 60 (19) 5494-8. Journal code: CNF. ISSN: 0008-5472.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200010
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20001027

AB The peroxisome proliferator-activated receptor gamma (PPARgamma) is a member of the **nuclear receptor** superfamily. Recent studies found that ligand-activated PPARgamma regulated differentiation and **clonal** growth of several types of cancer cells, including prostate cancer, suggesting that PPARgamma could be a tumor suppressor. Troglitazone was. . . reporter assays showed that the PPARgamma ligands troglitazone (10(-5) M), pioglitazone (10(-5) M), or 15-deoxy-delta12,14-prostaglandin J2 (10(-5) M) down-regulated androgen-stimulated **reporter gene** activity in LNCaP cells, a prostate cancer cell line. The PSA promoter contains androgen receptor response elements (AREs). **Reporter gene** studies showed that troglitazone inhibited androgen activation of the AREs in the PSA regulatory region. Consistent with inhibition of gene **expression**, 2 days of incubation of LNCaP with troglitazone dramatically suppressed PSA protein **expression** without suppressing AR **expression**, suggesting that troglitazone inhibited ARE activation by a mechanism other than down-regulation of **expression** of the AR. Taken together, ligands of PPARgamma may be a useful therapeutic approach for the treatment of prostate cancer. . .

L4 ANSWER 7 OF 33 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 2001132964 MEDLINE
DOCUMENT NUMBER: 21060441 PubMed ID: 10749678
TITLE: Characterization of a chicken retinoid X receptor-gamma gene promoter and identification of sequences that direct expression in retinal cells.
AUTHOR: Ameixa C; Brickell P M
CORPORATE SOURCE: Molecular Haematology Unit, Institute of Child Health, 30 Guilford Street, London WC1N 1EH, UK.
SOURCE: BIOCHEMICAL JOURNAL, (2000 Apr 15) 347 (Pt 2) 485-90. Journal code: 9YO; 2984726R. ISSN: 0264-6021.

PUB. COUNTRY: England: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-AJ239067
ENTRY MONTH: 200103
ENTRY DATE: Entered STN: 20010404
Last Updated on STN: 20010404
Entered Medline: 20010301

AB . . . the competence of cells to respond to extracellular signals. We have previously shown that, in the developing chick neural retina, **expression** of the retinoid X receptor-gamma (RXR-gamma2) **nuclear receptor** gene is restricted to photoreceptors. To characterize the mechanisms that regulate **expression** of this gene in the neural retina, we isolated a chicken RXR-gamma genomic **clone** containing the RXR-gamma2 promoter and mapped the transcription initiation site by means of ribonuclease protection. We analysed promoter activity by transient transfection of luciferase **reporter gene** constructs into cultured cells isolated from embryonic-chick neural retina or facial mesenchyme, which does not normally express detectable RXR-gamma2 transcripts.. . .

L4 ANSWER 8 OF 33 USPATFULL
ACCESSION NUMBER: 1999:117336 USPATFULL
TITLE: Specific expression vectors and methods of use
INVENTOR(S): Roop, Dennis R., Houston, TX, United States
Rothnagel, Joseph A., Houston, TX, United States
Greenhalgh, David A., Houston, TX, United States
PATENT ASSIGNEE(S): Baylor College of Medicine, Houston, TX, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5958764		19990928
APPLICATION INFO.:	US 1993-146930		19931101 (8)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1993-145388, filed on 29 Oct 1993, now abandoned which is a continuation-in-part of Ser. No. US 1992-876286, filed on 30 Apr 1992, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Chambers, Jasemine C.
ASSISTANT EXAMINER: Hauda, Karen M.
LEGAL REPRESENTATIVE: Lyon & Lyon LLP
NUMBER OF CLAIMS: 24
EXEMPLARY CLAIM: 1,20
NUMBER OF DRAWINGS: 11 Drawing Figure(s); 11 Drawing Page(s)
LINE COUNT: 2112
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM The 5' regulatory regions of four human epidermal keratin genes, K5, K6, K10 and K14, have been **cloned** into vectors to drive **expression** of the CAT **reporter gene**. These constructs were transfected into epithelial cells along with vectors expressing **nuclear receptors** for retinoic acid and thyroid hormone. (Tomic, et al., Cell Reg., Vol. 1, pp. 965-973 (1990)). This study demonstrated that. . . the human K1 gene responded to elevated levels of calcium in order to induce both mouse K1 and human K1 **expression**.

L4 ANSWER 9 OF 33 USPATFULL
ACCESSION NUMBER: 1999:85239 USPATFULL
TITLE: Methods and compositions for sensitive and rapid, functional identification of genomic polynucleotides

and secondary screening capabilities
INVENTOR(S): Whitney, Michael A., La Jolla, CA, United States
PATENT ASSIGNEE(S): Aurora Biosciences Corporation, San Diego, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5928888		19990727	
APPLICATION INFO.:	US 1996-719697		19960926	(8)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Fredman, Jeffrey
LEGAL REPRESENTATIVE: Fish & Richardson P.C.
NUMBER OF CLAIMS: 45
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 5 Drawing Figure(s); 4 Drawing Page(s)
LINE COUNT: 2581
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
DETD . . . cells will be separated by FACS. These two cell populations can
be treated with potential effectors and changes in gene **expression** monitored using ratio-metric fluorescent readout. Pools of **clones** will be isolated which show either up- or down-regulation of **reporter gene expression** . Target genes from responding **clones** can then be identified. In addition, by being able to separate expressing and non-expressing cells at different time points after. . . Specifically, it will provide a means to identify downstream genes which are transcriptionally
regulated by a variety of molecules including, **nuclear receptors**, cytokine receptors or transcription factors.

L4 ANSWER 10 OF 33 USPATFULL
ACCESSION NUMBER: 1999:69654 USPATFULL
TITLE: Keratin K1 expression vectors and methods of use
INVENTOR(S): Roop, Dennis R., Houston, TX, United States
Rothnagel, Joseph A., Houston, TX, United States
Greenhalgh, David A., Houston, TX, United States
Yuspa, Stuart H., Bethesda, MD, United States
PATENT ASSIGNEE(S): Baylor College of Medicine, Houston, TX, United States (U.S. corporation)
The United States of America as represented by the Department of Health and Human Services, Washington, DC, United States (U.S. government)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5914265		19990622	
APPLICATION INFO.:	US 1993-147777		19931101	(8)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1993-145387, filed on 29 Oct 1993, now abandoned which is a continuation-in-part of Ser. No. US 1992-876289, filed on 30 Apr 1992, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Chambers, Jasemine C.
ASSISTANT EXAMINER: Hauda, Karen M.
LEGAL REPRESENTATIVE: Lyon & Lyon LLP
NUMBER OF CLAIMS: 34
EXEMPLARY CLAIM: 1,21
NUMBER OF DRAWINGS: 10 Drawing Figure(s); 7 Drawing Page(s)
LINE COUNT: 3593
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
SUMM The 5' regulatory regions of four human epidermal keratin genes, K5, K6,
K10 and K14, have been **cloned** into vectors to drive

expression of the CAT **reporter gene**. These constructs were transfected into epithelial cells along with vectors expressing **nuclear receptors** for retinoic acid and thyroid hormone. (Tomic, et al., Cell Reg., Vol. 1, pp. 965-973 (1990)).

This study demonstrated that. . . the human K1 gene responded to elevated levels of calcium in order to induce both mouse K1 and human K1 **expression**.

L4 ANSWER 11 OF 33 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 1999289527 MEDLINE
DOCUMENT NUMBER: 99289527 PubMed ID: 10359768
TITLE: CPF: an orphan nuclear receptor that regulates liver-specific expression of the human cholesterol 7alpha-hydroxylase gene.
AUTHOR: Nitta M; Ku S; Brown C; Okamoto A Y; Shan B
CORPORATE SOURCE: Biology Department, Tularik Inc., Two Corporate Drive, South San Francisco, CA 94080, USA.
SOURCE: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (1999 Jun 8) 96 (12) 6660-5. Journal code: PV3; 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-AF146343
ENTRY MONTH: 199907
ENTRY DATE: Entered STN: 19990715
Last Updated on STN: 19990715
Entered Medline: 19990708

AB . . . this factor CPF, for CYP7A promoter binding factor. Mutation of the CPF binding site within the CYP7A promoter abolished hepatic-specific **expression** of the gene in transient transfection assays. A cDNA encoding CPF was **cloned** and identified as a human homolog of the Drosophila orphan **nuclear receptor** fushi tarazu F1 (Ftz-F1). Cotransfection of a CPF **expression** plasmid and a CYP7A **reporter gene** resulted in specific induction of CYP7A-directed transcription. These observations suggest that CPF is a key regulator of human CYP7A gene **expression** in the liver.

L4 ANSWER 12 OF 33 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:487454 CAPLUS
DOCUMENT NUMBER: 134:95896
TITLE: Molecular biology of hypoxia-inducible factor-1
AUTHOR(S): Wenger, Roland H.; Gassmann, Max
CORPORATE SOURCE: Institute of Physiology, University of Zurich-Irchel, Zurich, CH-8057, Switz.
SOURCE: Molecular Biology of Hematopoiesis 6, [Proceedings of the Symposium on the Molecular Biology of Hematopoiesis], 11th, Bormio, Italy, June 25-29, 1998 (1999), Meeting Date 1998, 269-276. Editor(s): Abraham, Nader G. Kluwer Academic/Plenum Publishers: New York, N. Y.
CODEN: 69ADIK
DOCUMENT TYPE: Conference; General Review
LANGUAGE: English
REFERENCE COUNT: 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

AB A review and discussion with 32 refs. The hypoxia-inducible factor-1 (HIF-1) is a basic-helix-loop-helix-PAS heterodimeric transcription factor that confers oxygen-regulated **expression** to a no. of genes

involved in oxygen homeostasis including erythropoietin (Epo), transferrin, glycolytic enzymes, and vascular endothelial growth factor (VEGF). Hypoxic exposure stabilizes the HIF-1.alpha. protein by a mechanism involving redox processes. Following heterodimerization with HIF-1.beta., better known as the aryl hydrocarbon **receptor nuclear** translocator (ARNT), HIF-1 binds to the DNA consensus sequence CGTG, known as a potential target of CpG methylation in mammalian cells. We showed that CpG methylation blocks HIF-1 DNA-binding as well as transactivation of **reporter gene expression**. The hypoxia-responsive 3' enhancer of the Epo gene was found to be methylation-free in Epo-producing cells despite its location outside of a CpG island. Intriguingly, this site was also methylation-free in cells that do not express Epo, indicating a general selective pressure to prevent CpG methylation, even in the absence of HIF-1 under normoxic conditions. We previously identified the constitutively expressed ATF-1/CREB-1 family members as candidate factors capable of binding the HIF-1 site. We **cloned** the mouse HIF-1.alpha. gene (designated Hif1a) and found that it consists of 15 exons dispersed over 45kb. Interestingly, mouse Hif1a contains two alternative first exons whose **expression** is driven by a tissue-specific promoter (exon I.1) or a house-keeping-type promoter located within a methylation-free CpG island (exon I.2). The exon I.1-contg. mRNA isoform encodes a predicted polypeptide that is 12 amino acids shorter than the exon I.2-derived mRNA isoform. So far, however, we did not find any functional differences between the two isoforms. The genomic Hif1a **clone** was. Used to introduce a null mutation into the mouse Hif1a locus by gene targeting in embryonic stem cells. HIF-1.alpha. deficiency is embryonic lethal, suggesting that HIF-1 serves as a non-redundant master regulator of oxygen homeostasis.

L4 ANSWER 13 OF 33 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:756786 CAPLUS
DOCUMENT NUMBER: 130:77022
TITLE: Mechanism of the interaction between orphan receptor TR3 and cis-acting element in ciliary neurotropic factor receptor CNTFR.alpha. gene
AUTHOR(S): Mu, Xiao-Min; Liu, Yi-Xun; Chang, Chawnshang
CORPORATE SOURCE: State Key Lab. Reproductive Biology, Inst. Zool., Chinese Acad. Sci., Beijing, 100080, Peop. Rep. China
SOURCE: Zhongguo Shengwu Huaxue Yu Fenzi Shengwu Xuebao (1998), 14(5), 485-491
CODEN: ZSHXF2; ISSN: 1007-7626
PUBLISHER: Zhongguo Shengwu Huaxue Yu Fenzi Shengwu Xuebao Bianweihui
DOCUMENT TYPE: Journal
LANGUAGE: Chinese
AB Ciliary neurotropic factor (CNTF) plays a very important role in the development and regeneration of the nervous system. CNTF utilizes a three-component receptor system mainly consisting of a CNTF-specific binding protein, known as CNTFR.alpha.. Orphan receptors is a category of receptors which cognate ligand is still unknown and belongs to **nuclear receptor** superfamily. TR3 (NGFI-B, Nur 77) is one of most important orphan receptors found so far. In order to investigate the interaction between TR3 and cis-acting elements in CNTFR.alpha. gene, the NBRE sequence of CNTFR.alpha.-15 was deleted by PCR using 2 pairs of synthesized oligonucleotide primers. The resulting two PCR fragments in the flank of the NBRE sequence were ligated and **cloned** into the EcoRV site of pT7 blue vector and then in the BglII site of pCAT-promoter vector, so **reporter genes** with NBRE-deleted CNTFR.alpha.-I5[CNTFR.alpha.-I5-NBRE(-)] inserting into pCAT-promotor vector with the orientation the same or opposite to CAT

reporter gene expression were constructed. The cell transfection and **reporter gene** assay using chloramphenicol acetyltransferase demonstrated that CNTFR.alpha.-I5-NBRE(-) still had an enhancer activity which could be induced by TR3 in a dose-dependent manner. The induction of CNTFR.alpha. gene **expression** by orphan receptor TR3 is not completely through the NBRE site, and other NBRE-like sequences in CNTFR.alpha.-I5 may also play some roles.

L4 ANSWER 14 OF 33 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:719276 CAPLUS
DOCUMENT NUMBER: 128:463
TITLE: Characteristics and function of the novel estrogen receptor .beta.
AUTHOR(S): Kuiper, George G. J.; Nilsson, Stefan; Gustafsson, Jan-Ake
CORPORATE SOURCE: Cent. Biotechnol. Dep. Med. Nutr., Karolinska Inst., Huddinge, Swed.
SOURCE: Horm. Signaling (1998), 1, 89-112
CODEN: HOSIFO
PUBLISHER: Academic
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review, with .apprx.100 refs. We have **cloned** a novel member of the **nuclear receptor** superfamily:estrogen receptor .beta. (ER.beta.). The cDNA of ER.beta. was isolated from a rat prostate cDNA library, and it encodes a protein of 485 amino acid residues with a calcd. mol. wt. of 54,200. The ER.beta. protein is highly homologous to the previously **cloned** ER.alpha. protein, particularly in the DNA-binding domain (95%) and in the C-terminal ligand-binding domain (55%). **Expression** of ER.beta. in rat tissues was investigated by in situ hybridization and RT-PCR; moderate to high **expression** was found in prostate (secretory epithelial cells), ovary (granulosa cells), lung, bladder, brain, uterus, epidydimis, and testis. Satn. ligand-binding anal. of in vitro-synthesized rat ER.beta. protein revealed a single binding component for 16.alpha.-iodo-3,17.beta.-estradiol with high affinity (Kd = 0.4 nM). In ligand-competition expts. the binding affinity decreased in the order dienestrol > 4-OH-tamoxifen > diethylstilbestrol > ICI-164384>17.beta.-estradiol > estrone > estriol > tamoxifen. In cotransfection expts. of Chinese hamster ovary cells with an ER.beta. **expression** vector and an estrogen-regulated **reporter gene**, maximal stimulation of **reporter gene** activity was found during incubation with 1 nM 17.beta.-estradiol. The detailed biol. significance of the existence of two different RERs is at this moment unclear. Differences in the ligand-binding properties and/or transactivation function on certain target genes may exist.

L4 ANSWER 15 OF 33 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:638138 CAPLUS
DOCUMENT NUMBER: 130:33079
TITLE: Cloning, expression and function of a novel estrogen receptor
AUTHOR(S): Kuiper, George G. J. M.; Nilsson, Stefan; Gustafsson, Jan-Ake
CORPORATE SOURCE: Center for Biotechnology and Department of Medical Nutrition, Karolinska Institute, Huddinge, S-14186, Swed.
SOURCE: Endothelial Cell Res. Ser. (1998), 3(Estrogen and the Vessel Wall), 1-17
CODEN: ECRSFY; ISSN: 1384-1270
PUBLISHER: Harwood Academic Publishers
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

REFERENCE COUNT: 68 THERE ARE 68 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

AB A review, with 68 refs. We have **cloned** a novel member of the **nuclear receptor** superfamily; estrogen receptor .beta. (ER.beta.). The cDNA of ER.beta. was isolated from a rat prostate cDNA library and it encodes a protein of 485 amino acid residues with a calcd. mol. wt. of 54200. The ER.beta. protein is highly homologous to the previously **cloned** ER.alpha. protein, particularly in the DNA-binding domain (95%) and in the C-terminal ligand-binding domain (55%). **Expression** of ER.beta. in rat tissues was investigated by in situ hybridization and RT-PCR; moderate to high **expression** was found in prostate (secretory epithelial cells), ovary (granulosa cells), lung, bladder, brain, uterus and testis. Satn. ligand-binding anal. of in vitro synthesized rat ER.beta. protein revealed a single binding component for 16.alpha.-iodo-3,17.beta.-estradiol with high affinity (Kd = 0.4 nM). In ligand-competition expts. the binding affinity decreased in the order dienestrol > 4-OH-tamoxifen > diethylstilbestrol > ICI-164384 > 17.beta.-estradiol > estrone > estriol > tamoxifen. In co-transfection expts. of Chinese hamster ovary cells with an ER.beta. **expression** vector and an estrogen-regulated **reporter gene**, maximal stimulation of **reporter gene** activity was found during incubation with 1 nM of 17.beta.-estradiol. The detailed biol. significance of the existence of two different ERs is at this moment unclear. Differences in the ligand-binding properties and/or transactivation function on certain target genes may exist.

L4 ANSWER 16 OF 33 MEDLINE DUPLICATE 5
ACCESSION NUMBER: 97165873 MEDLINE
DOCUMENT NUMBER: 97165873 PubMed ID: 9013766
TITLE: RIP 140 enhances nuclear receptor-dependent transcription in vivo in yeast.
AUTHOR: Joyeux A; Cavailles V; Balaguer P; Nicolas J C
CORPORATE SOURCE: INSERM U439, Montpellier, France.
SOURCE: MOLECULAR ENDOCRINOLOGY, (1997 Feb) 11 (2) 193-202.
Journal code: NGZ; 8801431. ISSN: 0888-8809.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199705
ENTRY DATE: Entered STN: 19970609
Last Updated on STN: 19970609
Entered Medline: 19970528

AB RIP140 has previously been **cloned** as a factor that interacts with the estrogen receptor (ER) in vitro. We demonstrate in this study that RIP140 is a co-factor for **nuclear receptor** in yeast. RIP140 enhances the ER transcriptional activity by increasing 1.5- to 4-fold the induction factor of the **reporter gene** response at saturating hormone concentrations, this effect being magnified at suboptimal doses of estradiol. Moreover, RIP140 decreases the ED50 of. . . the AF2-AD domain and in a agonist-dependent fashion. RIP140 is also a strong coactivator for the retinoid pathway, as its **expression** enhances 10-fold the transactivation of a chimeric retinoic acid-alpha receptor at saturant hormone concentration and left shifted 5-fold the ED50. . .

L4 ANSWER 17 OF 33 MEDLINE DUPLICATE 6
ACCESSION NUMBER: 1998109011 MEDLINE
DOCUMENT NUMBER: 98109011 PubMed ID: 9447705
TITLE: The zebrafish thyroid hormone receptor alpha 1 is expressed

during early embryogenesis and can function in transcriptional repression.
AUTHOR: Essner J J; Breuer J J; Essner R D; Fahrenkrug S C; Hackett P B Jr
CORPORATE SOURCE: Department of Genetics and Cell Biology, University of Minnesota, St. Paul 55108-1095, USA.
CONTRACT NUMBER: RO1-RR06625 (NCRR)
SOURCE: DIFFERENTIATION, (1997 Dec) 62 (3) 107-17.
Journal code: E99; 0401650. ISSN: 0301-4681.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-U54796
ENTRY MONTH: 199802
ENTRY DATE: Entered STN: 19980226
Last Updated on STN: 19980226
Entered Medline: 19980213

AB **Nuclear receptors** are a large family of ligand dependent transcription factors which participate in many diverse processes during development. In this report, we describe the **cloning** of the zebrafish thyroid hormone receptor alpha 1 (TR alpha 1) gene, the cellular counterpart of the viral oncogene v-erbA.. . . to the embryo. TR alpha 1 is expressed again after the mid blastula transition. By examining the effects of increased **expression** of TR alpha 1 on **expression** of a **reporter gene** which responds to both TR alpha 1 and retinoic acid receptors (RARs), we show that the zebrafish TR alpha 1. . .

L4 ANSWER 18 OF 33 MEDLINE DUPLICATE 7
ACCESSION NUMBER: 1998005040 MEDLINE
DOCUMENT NUMBER: 98005040 PubMed ID: 9344589
TITLE: Stable transfection of U937 cells with sense or antisense RXR-alpha cDNA suggests a role for RXR-alpha in the control of monoblastic differentiation induced by retinoic acid and vitamin D.
AUTHOR: Brown T R; Stonehouse T J; Branch J S; Brickell P M; Katz D R
CORPORATE SOURCE: Department of Molecular Pathology, University College London Medical School, United Kingdom.
SOURCE: EXPERIMENTAL CELL RESEARCH, (1997 Oct 10) 236 (1) 94-102.
Journal code: EPB; 0373226. ISSN: 0014-4827.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199711
ENTRY DATE: Entered STN: 19971224
Last Updated on STN: 19971224
Entered Medline: 19971125

AB . . . monoblast lineage. In particular, the part played by the retinoid X receptors (RXRs), which are members of the steroid/thyroid hormone **nuclear receptor** family, has not been explored. In this study, therefore, the human monoblastic leukemia cell line U937 has been used as. . . lines which expressed either increased or decreased levels of RXR-alpha, respectively. The sense cell lines (U alpha S and its **clonal** derivative alpha G2S) showed increased sensitivity to RA, while the antisense cell lines (U alpha A and its **clonal** derivative alpha B5A) showed decreased sensitivity to RA, as demonstrated

by growth inhibition and by regulation of an RA-responsive **reporter gene**. Both U alpha A and alpha B5A also failed to respond to another modulating agent, 1 alpha,25-dihydroxycholecalciferol (DHCC), but only. . . of RA and DHCC together inhibited growth of both sense and antisense cell lines. In addition, alpha G2S exhibited increased **expression** of CD11b and CD54, while alpha B5A cells showed increased **expression** of CD102, suggesting that RXR-alpha has a role in regulating **expression** of cell adhesion molecules in U937 cells. These results demonstrate that RXR-alpha has a role in mediating growth inhibition and. . .

L4 ANSWER 19 OF 33 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE
8
ACCESSION NUMBER: 1998:120943 BIOSIS
DOCUMENT NUMBER: PREV199800120943
TITLE: Introduction of exogenous thyroid hormone receptors modifies growth hormone gene expression in GH3 cell.
AUTHOR(S): Hayashi, Yoshitaka (1); Shibata, Taiga; Ito, Takeshi; Murata, Yoshiharu; Seo, Hisao
CORPORATE SOURCE: (1) Dep. Endocrinol. and Metabolism, Div. Molecular and Cellular Adaptation Research Inst. Environmental Med., Nagoya Univ., Furo-cho, Chikusa-ku, Nagoya 464-01 Japan
SOURCE: Environmental Medicine (Nagoya), (Dec., 1997) Vol. 41, No. 2, pp. 83-85.
ISSN: 0287-0517.
DOCUMENT TYPE: Article
LANGUAGE: English
AB **Cloning** of nuclear hormone receptor cDNAs and identification of hormone responsive elements which mediate hormonal action enabled the study of hormonal control of gene **expression** at the molecular level. In these studies the effect of overexpression of hormone receptors is mainly analyzed using artificial hormone-responsive **reporter genes**. In the present report, we studied how overexpression of nuclear hormone receptors using recombinant adenoviral vectors modifies endogenous growth hormone gene **expression** in GH3 cells. Growth hormone mRNA in GH3 cells not infected with recombinant virus was increased 2.13 +- 0.53-fold by. . . the triiodothyronine-mediated increase to 5.45 +- 2.13-fold. Since endogenous genes have a complex chromatin structure which is absent in artificial **reporter genes**, the present system is useful in studying the physiological action of **nuclear receptors**.

L4 ANSWER 20 OF 33 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1996:551368 CAPLUS
DOCUMENT NUMBER: 125:214277
TITLE: Method for identifying RXR-interacting proteins (RIP's) and sequences of RIP's and RIP cDNA's
INVENTOR(S): Moore, David; Seol, Wongi; Choi, Hueng-Sik
PATENT ASSIGNEE(S): General Hospital Corporation, USA
SOURCE: PCT Int. Appl., 79 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9621677	A1	19960718	WO 1995-US16311	19951208
W: JP				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
US 5932699	A	19990803	US 1995-372652	19950113
EP 801657	A1	19971022	EP 1995-943114	19951208
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, PT, IE				

PRIORITY APPLN. INFO.: US 1995-372652 19950113

AB Disclosed is a method for detg. whether a test protein, is capable of interacting with a retinoid X receptor protein. The method involves: (a) providing a host cell which contains (i) a **reporter gene** operably linked to a protein binding site; (ii) a first fusion gene which expresses a first fusion protein, the first fusion protein including a retinoid X receptor protein covalently bonded to a binding moiety which is capable of specifically binding to the protein binding site; and (iii) a second fusion gene which expresses a second fusion protein, the second fusion protein including the test protein covalently bonded to a gene activating moiety; and (b) detg. whether the test protein increases **expression** of the **reporter gene** as an indication of its ability to interact with the retinoid X receptor protein. Also disclosed is purified DNA encoding retinoid X receptor-interacting proteins (RIP's) and the polypeptides expressed from such DNA. The interaction trap technique was used to isolate cDNA's encoding proteins that interact with the ligand-binding domain of human RXR.alpha.. Two **clones**, RIP14 and RIP 15, were previously undescribed orphan members of the **nuclear receptor** superfamily while two others showed no significant similarity to any known protein and are candidate transcriptional coactivators. **Expression** of RIP genes in various tissues, binding of the RIP's to other receptors and binding to DNA was examd. RIP14 and RIP15 bound to an overlapping set of specific elements (e.g EcRE and .beta.RARE) as heterodimers with RXR.alpha..

L4 ANSWER 21 OF 33 MEDLINE DUPLICATE 9

ACCESSION NUMBER: 97113002 MEDLINE
DOCUMENT NUMBER: 97113002 PubMed ID: 8943255
TITLE: Characterization of the promoter of the rat sarcoplasmic endoplasmic reticulum Ca2+-ATPase 1 gene and analysis of thyroid hormone responsiveness.
AUTHOR: Simonides W S; Brent G A; Thelen M H; van der Linden C G; Larsen P R; van Hardeveld C
CORPORATE SOURCE: Thyroid Division, Brigham and Women's Hospital, Department of Medicine, Harvard Medical School, Boston, Massachusetts 02115, USA.. ws.simonides.physiol@med.vu.nl
CONTRACT NUMBER: DK 44128 (NIDDK)
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1996 Dec 13) 271 (50) 32048-56.
Journal code: HIV; 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-U34282
ENTRY MONTH: 199701
ENTRY DATE: Entered STN: 19970219
Last Updated on STN: 19970219
Entered Medline: 19970117

AB . . . muscle requires the re-uptake of Ca2+, which is mediated by the sarcoplasmic reticulum Ca2+-ATPase (SERCA). Thyroid hormone (T3) stimulates the **expression** of the SERCA1 isoform, which is essential for fast skeletal muscle fiber phenotype. We have **cloned** and studied the first 962 base pairs of the 5'-flanking region of the rat SERCA1 gene. This sequence was tested for T3-regulated **expression** in transient transfection experiments using COS7 cells and for binding of thyroid hormone receptor (TR) alpha in mobility shift assays. A construct of the 5'-flanking region and a **reporter gene** was unresponsive to T3 in the absence of co-transfected thyroid hormone receptor. In the presence of TRalpha, a T3 induction ratio of almost 4.0 was found, and this induction ratio was doubled with co-transfection of an

RXR **expression** plasmid. Analysis of progressive 5'-deletion fragments of the sequence indicated multiple regions involved in T3 responsiveness. Three regions, R1, R2,. . . half-sites, comprising two independent thyroid hormone response elements, interact cooperatively to give the maximal T3 response. T3 regulation of SERCA1 **expression** is mediated by a complex thyroid hormone response element that may serve to provide a greater range of response in interaction with **nuclear receptor** partners or cell-specific transcription factors.

L4 ANSWER 22 OF 33 MEDLINE DUPLICATE 10
ACCESSION NUMBER: 96234066 MEDLINE
DOCUMENT NUMBER: 96234066 PubMed ID: 8650195
TITLE: Cloning of a novel receptor expressed in rat prostate and ovary.
AUTHOR: Kuiper G G; Enmark E; Pelto-Huikko M; Nilsson S; Gustafsson J A
CORPORATE SOURCE: Center for Biotechnology and Department of Medical Nutrition, Karolinska Institute, Huddinge, Sweden.
SOURCE: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (1996 Jun 11) 93 (12) 5925-30. Journal code: PV3; 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-U57439
ENTRY MONTH: 199607
ENTRY DATE: Entered STN: 19960805
Last Updated on STN: 19960805
Entered Medline: 19960725

AB We have **cloned** a novel member of the **nuclear receptor** superfamily. The cDNA of **clone** 29 was isolated from a rat prostate cDNA library and it encodes a protein of 485 amino acid residues with a calculated molecular weight of 54.2 kDa. **Clone** 29 protein is unique in that it is highly homologous to the rat estrogen receptor (ER) protein, particularly in the DNA-binding domain (95%) and in the C-terminal ligand-binding domain (55%). **Expression** of **clone** 29 in rat tissues was investigated by in situ hybridization and prominent **expression** was found in prostate and ovary. In the prostate **clone** 29 is expressed in the epithelial cells of the secretory alveoli, whereas in the ovary the granuloma cells in primary, secondary, and mature follicles showed **expression** of **clone** 29. Saturation ligand-binding analysis of in vitro synthesized **clone** 29 protein revealed a single binding component for 17beta-estradiol (E2) with high affinity (Kd= 0.6 nM). In ligand-competition experiments the. . . > 5alpha-androstane-3beta,17beta-diol >> testosterone = progesterone = corticosterone = 5alpha-androstane-3alpha,17beta-diol. In cotransfection experiments of Chinese hamster ovary cells with a **clone** 29 **expression** vector and an estrogen-regulated **reporter gene**, maximal stimulation (about 3-fold) of **reporter gene** activity was found during incubation with 10 nM of E2. Neither progesterone, testosterone, dexamethasone, thyroid hormone, all-trans-retinoic acid, nor 5alpha-androstane-3alpha,I7beta-diol could stimulate **reporter gene** activity, whereas estrone and 5alpha-androstane-3beta,17beta-diol did. We conclude that **clone** 29 cDNA encodes a novel rat ER, which we suggest be named rat ERbeta to distinguish it from the previously **cloned** ER (ERalpha) from rat uterus.

L4 ANSWER 23 OF 33 MEDLINE DUPLICATE 11
ACCESSION NUMBER: 96192924 MEDLINE
DOCUMENT NUMBER: 96192924 PubMed ID: 8614404

TITLE: TOR: a new orphan receptor expressed in the thymus that can modulate retinoid and thyroid hormone signals.
AUTHOR: Ortiz M A; Piedrafita F J; Pfahl M; Maki R
CORPORATE SOURCE: La Jolla Cancer Research Foundation, California 92037, USA.
SOURCE: MOLECULAR ENDOCRINOLOGY, (1995 Dec) 9 (12) 1679-91.
Journal code: NGZ; 8801431. ISSN: 0888-8809.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-U39071
ENTRY MONTH: 199606
ENTRY DATE: Entered STN: 19960613
Last Updated on STN: 19960805
Entered Medline: 19960603

AB . . . of vitamin A, retinoic acid, as well as vitamin D3 and thyroid hormones exert their actions by binding to specific **nuclear receptors** that represent one subfamily of the steroid/thyroid hormone receptor superfamily. To identify new members of the retinoid/thyroid hormone receptor subfamily. . . the immune system, a screening of a T cell cDNA library was performed using a retinoid X receptor probe. A **clone** was isolated encoding a novel **nuclear receptor** expressed mainly in the thymus and T cell line s. This new receptor, TOR (thymus orphan receptor), is most closely. . . two receptors and RZR beta in a phylogenetic tree, when both the DNA-binding domain and the ligand-binding domain sequences of **nuclear receptors** are compared. Thus, TOR is part of a subgroup of receptors, one of which has recently been reported to be. . . binding sites for thyroid hormone (TR), and retinoic acid receptors (RAR). In transient transfection experiments TOR does not activate a **reporter gene** carrying these sequences in the absence or the presence of any known **nuclear receptor** ligands. TOR, however, is able to repress TR and RAR activity on DR-4-TREs or DR-5-RAREs, respectively. Therefore, our data suggest. . . regulate retinoic acid and thyroid hormone signals. However, the response elements recognized by TOR and COUP-TF differ as do the **expression** patterns of these receptors. Thus, one important role of TOR could be to modulate retinoid and thyroid hormone signals in. . .

L4 ANSWER 24 OF 33 MEDLINE DUPLICATE 12
ACCESSION NUMBER: 96062010 MEDLINE
DOCUMENT NUMBER: 96062010 PubMed ID: 7488247
TITLE: Functional analysis of aryl hydrocarbon receptor nuclear translocator interactions with aryl hydrocarbon receptor in the yeast two-hybrid system.
AUTHOR: Yamaguchi Y; Kuo M T
CORPORATE SOURCE: Department of Molecular Pathology, University of Texas M.D. Anderson Cancer Center, Houston 77030, USA.
CONTRACT NUMBER: CA55813 (NCI)
CA55846 (NCI)
SOURCE: BIOCHEMICAL PHARMACOLOGY, (1995 Oct 12) 50 (8) 1295-302.
Journal code: 9Z4; 0101032. ISSN: 0006-2952.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199512
ENTRY DATE: Entered STN: 19960124
Last Updated on STN: 19970203
Entered Medline: 19951214

AB . . . receptor (AHR) mediates dioxin (2,3,7,8-tetrachlorodibenzo-p-dioxin)-induced transcriptional activation of a battery of genes by

interaction with a cofactor, called aryl hydrocarbon **receptor nuclear** translocator (ARNT) protein. Both AHR and ARNT belong to a family of proteins that includes the Drosophila circadian-rhythm protein and. . . which contains the bHLH and PAS regions, to screen cDNA libraries prepared from human lymphocytes and C57BL mouse liver for **clones** encoding proteins capable of binding to these regions, we isolated a partial ARNT cDNA **clone**. These results demonstrated that the N-terminal half of AHR is capable of interacting with ARNT in yeast (probably through the. . . A fusion protein containing the GAL4 DNA binding domain (DB) linked to the full-length AHR was not capable of activating **expression** of a **reporter gene** containing the GAL4 DNA binding site, suggesting that ligand-free AHR alone has no transactivating properties in yeast. However, the C-terminal portion (amino acid residues 580-797) of the AHR, including the Q-rich domain, could confer transactivation of the **reporter gene expression** in the same system, suggesting that the N-terminal portion of the AHR contains transcription repression properties. In contrast, GAL4(DB)-ARNT fusion protein was able to activate **expression** of the same **reporter gene**. Deletion analysis of ARNT revealed that the C-terminal 75 amino acids, including the Q-rich domain, exhibited full transactivation function in. . .

L4 ANSWER 25 OF 33 MEDLINE DUPLICATE 13
ACCESSION NUMBER: 95352492 MEDLINE
DOCUMENT NUMBER: 95352492 PubMed ID: 7626496
TITLE: Fatty acid activation of peroxisome proliferator-activated receptor (PPAR).
AUTHOR: Bocos C; Gottlicher M; Gearing K; Banner C; Enmark E; Teboul M; Crickmore A; Gustafsson J A
CORPORATE SOURCE: Department of Medical Nutrition, Karolinska Institute, Huddinge University Hospital, Sweden.
SOURCE: JOURNAL OF STEROID BIOCHEMISTRY AND MOLECULAR BIOLOGY, (1995 Jun) 53 (1-6) 467-73. Ref: 40
Journal code: AX4; 9015483. ISSN: 0960-0760.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199509
ENTRY DATE: Entered STN: 19950921
Last Updated on STN: 19970203
Entered Medline: 19950907
AB . . . as clofibric acid, nafenopin, and WY-14,643 have been shown to activate peroxisome proliferator-activated receptor (PPAR), a member of the steroid **nuclear receptor** superfamily. We have **cloned** the cDNA from rat that is homologous to that from mouse, which encodes a 97% similar protein. To search for. . . transactivation assay by stably expressing in CHO cells a chimera of rat PPAR and the human glucocorticoid receptor that activates **expression** of the placental alkaline phosphatase **reporter gene** under the control of the mouse mammary tumor virus promoter. 150 microM concentrations of arachidonic or linoleic acid but not of dehydroepiandrosterone, cholesterol, or 25-hydroxy-cholesterol, activated the receptor chimera. In addition, saturated fatty acids induced the **reporter gene**. Shortening the chain length to n = 6 or introduction of an omega-terminal carboxylic group abolished the activation potential of. . .

L4 ANSWER 26 OF 33 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1995:487761 CAPLUS
DOCUMENT NUMBER: 123:2402
TITLE: Assignment of the human ubiquitous receptor gene (UNR)

to 19q13.3 using fluorescence in situ hybridization
AUTHOR(S): Le Beau, Michelle M.; Song, Ching; Davis, Elizabeth M.; Hiipakka, Richard A.; Kokontis, John M.; Liao, Shutsung
CORPORATE SOURCE: Dep. Med., Univ. Chicago, Chicago, IL, 60637, USA
SOURCE: Genomics (1995), 26(1), 166-8
CODEN: GNMCEP; ISSN: 0888-7543
DOCUMENT TYPE: Journal
LANGUAGE: English

AB We recently **cloned** the human and rat cDNAs for a new member of the **nuclear receptor** family, which we named ubiquitous receptor (UR) because ofits **expression** in many tissues. The symbol for this gene is UNR (ubiquitous **nuclear receptor**). UR is a 50-kDa nuclear protein that belongs to the thyroid hormone/retinoic acid receptor subfamily of **nuclear receptors**, based on the P-box amino acids of its DNA-binding domain and its ability to bind to AGGTCA direct repeats with four-nucleotide (DR4) spacing as a heterodimer with RXR. In the absence of 9-cis-retinoic acid, coexpression of UR in combination with RXR in COS-1 cells stimulated a **reporter gene** contg. a DH4 response element. It is not known whether a ligand is required for UR function. Coexpression of UR inhibited RAR and RXR activation of DR4-linked **reporter gene expression**, but not a DR5-linked **reporter gene**, in the presence of all-trans-retinoic acid. Since UR can modulate the retinoid and thyroid hormone signaling pathways, it may have an important role in normal growth and differentiation. Human UNR cDNAs were used to screen a Lambda FIX II human male placenta genomic library (Stratagene). Phage DNA from **clones** hybridizing to UNR cDNA was characterized by Southern hybridization and restriction mapping, and two different **clones** (hG10 and hG12) with inserts of 15-20 kb were chosen for fluorescence in situ hybridization (FISH) anal. Biotin-labeled probes were prepd. from phage DNA by nick-translation using Bio-11-dUTP (Enzo Diagnostics). FISH was performed as described previously. Hybridization was detected with fluorescein-conjugated avidin (Vector Labs.), and chromosomes were identified by staining with 4,6-diamidino-2-phenylindole-dihydrochloride (DAPI). Hybridization of the UNR probe to normal human metaphase chromosomes resulted in specific labeling only of chromosome 19. Specific labeling of 19q13 was obsd. on four (14 cells), three (6 cells), two (4 cells), or one (1 cell) chromatid(s) of the chromosome 19 homologs in 25 cells examd. Of 85 signals obsd. (83 of 100 19q chromatids from 25 metaphase cells were labeled), 83 (97.6%) were located at 19q13.3. The remaining 2 signals were located at 17q25 (2.4%). Specific labeling of 19q13.3 was obtained in an addnl. hybridization expt. using the hG10 probe and in other hybridizations using another probe (hG12) for this gene. These results indicate that the UNR gene is localized to chromosome 19q13.3.

L4 ANSWER 27 OF 33 MEDLINE DUPLICATE 14
ACCESSION NUMBER: 95062154 MEDLINE
DOCUMENT NUMBER: 95062154 PubMed ID: 7971966
TITLE: Ubiquitous receptor: a receptor that modulates gene activation by retinoic acid and thyroid hormone receptors.
AUTHOR: Song C; Kokontis J M; Hiipakka R A; Liao S
CORPORATE SOURCE: Ben May Institute, Chicago, IL.
CONTRACT NUMBER: CA58073 (NCI)
DK37694 (NIDDK)
DK41670 (NIDDK)
SOURCE: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (1994 Nov 8) 91 (23) 10809-13.
Journal code: PV3; 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States

Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-U14533; GENBANK-U14534
ENTRY MONTH: 199412
ENTRY DATE: Entered STN: 19950110
Last Updated on STN: 19950110
Entered Medline: 19941212

AB The cDNA for a member of the **nuclear receptor** family was **cloned** and named ubiquitous receptor (UR), since UR protein and mRNA are detected in many cell types. Rat UR/human retinoid X. . . half-site sequence AGGTCA and 4-nt spacing (DR-4). Coexpression of UR in COS-1 cells inhibited the stimulation of chloramphenicol acetyltransferase (CAT) **reporter gene expression** by hRXR alpha and human retinoic acid receptor alpha in the presence of all-trans-retinoic acid when DR-4 (but not DR-5) was present upstream of the promoter of a CAT **reporter gene** (DR-4-CAT). UR **expression** also inhibited the activation of a DR-4-CAT **reporter gene** by hRXR alpha and 9-cis-retinoic acid or by thyroid hormone receptor beta in the presence of thyroid hormone. However, in the absence of 9-cis-retinoic acid, UR in combination with hRXR alpha stimulation DR-4-CAT **expression**. Coexpression of thyroid hormone receptor markedly reduced this stimulation in the absence of thyroid hormone. UR may play an important. . .

L4 ANSWER 28 OF 33 MEDLINE DUPLICATE 15
ACCESSION NUMBER: 94221603 MEDLINE
DOCUMENT NUMBER: 94221603 PubMed ID: 8168101
TITLE: Antiestrogenic effect of 2,3,7,8-tetrachlorodibenzo-p-dioxin on 17 beta-estradiol-induced pS2 expression.
AUTHOR: Zacharewski T R; Bondy K L; McDonell P; Wu Z F
CORPORATE SOURCE: Department of Pharmacology and Toxicology, University of Western Ontario, London, Canada.
SOURCE: CANCER RESEARCH, (1994 May 15) 54 (10) 2707-13.
Journal code: CNF; 2984705R. ISSN: 0008-5472.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199406
ENTRY DATE: Entered STN: 19940613
Last Updated on STN: 19980206
Entered Medline: 19940602

AB . . . decreased E2-induced secreted pS2 protein levels by 50% and the induction of the transiently transfected -1100 to -86 pS2 promoter-regulated **reporter gene** (pS2-LUC) by 57%. Comparable effects on PS2-LUC activity were observed in HeLa and ZR-75 cells. In contrast, TCDD had minimal. . . induction, whereas treatment with 10 nM ICI 164,384 caused a 60% decrease in luciferase activity. In Hepa 1c1c7 wild-type and **clone** 1 (C1) mutant cells, TCDD also reduced E2 induction of pS2-LUC activity but had little effect in **clone** 4 (C4) or **clone** 12 (C12) mutant cells. However, suppression was reestablished following transfection of the human Ah **receptor nuclear** translocator (ARNT) complementary DNA **expression** vector into C4 cells and the mouse Ah receptor (AhR) complementary DNA **expression** vector into C12 cells. Induction of pS2-LUC activity by the ligand-dependent and -independent chimeric estrogen receptors (HE15, HE19, ERcVP16, and. . . effective (38 and 20%, respectively). These results demonstrate a role for the Ah receptor in TCDD-mediated suppression of E2-induced pS2 **expression**. Data is presented demonstrating that the effect requires sequences within the pS2 promoter other than the estrogen response element and. . .

L4 ANSWER 29 OF 33 MEDLINE DUPLICATE 16
ACCESSION NUMBER: 95140028 MEDLINE

DOCUMENT NUMBER: 95140028 PubMed ID: 7838156
TITLE: Identification of RVR, a novel orphan nuclear receptor that
acts as a negative transcriptional regulator.
AUTHOR: Retnakaran R; Flock G; Giguere V
CORPORATE SOURCE: Division of Endocrinology, Hospital for Sick Children, Toronto, Canada.
SOURCE: MOLECULAR ENDOCRINOLOGY, (1994 Sep) 8 (9) 1234-44.
Journal code: NGZ; 8801431. ISSN: 0888-8809.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-U12142
ENTRY MONTH: 199502
ENTRY DATE: Entered STN: 19950314
Last Updated on STN: 19950314
Entered Medline: 19950227

AB A novel member of the steroid/thyroid/retinoid superfamily of **nuclear receptors** has been isolated as part of a screen to identify genes related to the recently characterized orphan receptor ROR alpha. This new orphan receptor, **cloned** from a mouse brain cDNA library, is closely related to the rat Rev-ErbA alpha gene product (97% and 68% identity. . . it binds the DNA sequence ATAACTAGGTCA, a hormone response element composed of a 6-base pair AT-rich sequence preceding a single **nuclear receptor** recognition half-site core motif PuGGTCA. We show that RVR recognizes this hormone response element with a specificity similar to that. . . 2. However, cotransfection studies indicate that RVR does not activate transcription when this hormone response element is linked to a **reporter gene** but rather acts as a potent competitive repressor of ROR alpha function. These results indicate the existence of an orphan **nuclear receptor**-based signaling pathway with the intrinsic ability to regulate the **expression** of specific gene networks through competition between transcriptional activators and repressors for the same recognition site.

L4 ANSWER 30 OF 33 MEDLINE DUPLICATE 17
ACCESSION NUMBER: 94364428 MEDLINE
DOCUMENT NUMBER: 94364428 PubMed ID: 8082729
TITLE: A beta 2RARE-LacZ transgene identifies retinoic acid-mediated transcriptional activation in distinct cutaneous sites.
AUTHOR: Tsou H C; Si S P; Lee X; Gonzalez-Serva A; Peacocke M
CORPORATE SOURCE: Department of Dermatology, New England Medical Center, Boston, Massachusetts 02111.
CONTRACT NUMBER: AG-09927 (NIA)
SOURCE: EXPERIMENTAL CELL RESEARCH, (1994 Sep) 214 (1) 27-34.
Journal code: EPB; 0373226. ISSN: 0014-4827.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199410
ENTRY DATE: Entered STN: 19941021
Last Updated on STN: 19970203
Entered Medline: 19941012

AB . . . in a variety of tissues, including the skin. How retinoic acid mediates these effects is not fully understood. The recent **cloning** of a series of **nuclear receptors** for retinoic acid (RARs) has demonstrated that these proteins can function as ligand-inducible transcriptional enhancing factors. Moreover, all receptors are members of the steroid/thyroid hormone multigene family. In vitro studies have demonstrated the **expression** of RAR alpha, RAR beta, and RAR gamma in various cell types found in the skin. While multiple isoforms exist. . . model in which the retinoic acid response

element (RARE) of the RAR beta 2 isoform is linked to a beta-galactosidase **reporter gene**. Our observations consistently demonstrate that retinoic acid transcriptionally activates the beta 2RARE in distinct areas of the skin. Of interest,. . .

L4 ANSWER 31 OF 33 MEDLINE DUPLICATE 18
ACCESSION NUMBER: 92262498 MEDLINE
DOCUMENT NUMBER: 92262498 PubMed ID: 1316614
TITLE: Fatty acids activate a chimera of the clofibric acid-activated receptor and the glucocorticoid receptor.
AUTHOR: Gottlicher M; Widmark E; Li Q; Gustafsson J A
CORPORATE SOURCE: Department of Medical Nutrition, Karolinska Institute, Huddinge, Sweden.
SOURCE: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (1992 May 15) 89 (10) 4653-7. Journal code: PV3; 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-M88592
ENTRY MONTH: 199206
ENTRY DATE: Entered STN: 19920626
Last Updated on STN: 19970203
Entered Medline: 19920616

AB . . . as clofibric acid, nafenopin, and WY-14,643 have been shown to activate PPAR (peroxisome proliferator-activated receptor), a member of the steroid **nuclear receptor** superfamily. We have **cloned** the cDNA from the rat that is homologous to that from the mouse [Issemann, I. & Green, S. (1990) Nature. . . transactivation assay by stably expressing in CHO cells a chimera of rat PPAR and the human glucocorticoid receptor that activates **expression** of the placental alkaline phosphatase **reporter gene** under the control of the mouse mammary tumor virus promoter. Testing of compounds related to lipid metabolism or peroxisomal proliferation. . . linoleic acid but not of dehydroepiandrosterone, cholesterol, or 25-hydroxy-cholesterol, activate the receptor chimera. In addition, saturated fatty acids induce the **reporter gene**. Shortening the chain length to n = 6 or introduction of an omega-terminal carboxylic group abolished the activation potential of the fatty acid. In conclusion, the present results indicate that fatty acids can regulate gene **expression** mediated by a member of the steroid **nuclear receptor** superfamily.

L4 ANSWER 32 OF 33 MEDLINE DUPLICATE 19
ACCESSION NUMBER: 93187145 MEDLINE
DOCUMENT NUMBER: 93187145 PubMed ID: 1284070
TITLE: Regulation of epidermal keratin expression by retinoic acid and thyroid hormone.
AUTHOR: Ohtsuki M; Tomic-Canic M; Freedberg I M; Blumenberg M
CORPORATE SOURCE: Ronald O Perelman Department of Dermatology, New York University Medical Center, New York 10016.
CONTRACT NUMBER: AR30682 (NIAMS)
AR39176 (NIAMS)
DK16636 (NIDDK)
+
SOURCE: JOURNAL OF DERMATOLOGY, (1992 Nov) 19 (11) 774-80. Journal code: HZ7; 7600545. ISSN: 0385-2407.
PUB. COUNTRY: Japan
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199304
ENTRY DATE: Entered STN: 19930416

Last Updated on STN: 19960129
Entered Medline: 19930405

AB In the epidermis, retinoic acid (RA) is known to regulate the gene **expression** of keratins, the intermediate filament proteins of epithelial cells. We have **cloned** the upstream regulatory regions of three human epidermal keratin genes, K5, K10, and K14, and engineered DNA constructs in which these regions drive **expression** of the CAT **reporter gene**. By co-transfecting the constructs into various epithelial cell types along with the vectors expressing the **nuclear receptors** for RA and thyroid hormone (T3), we have shown that RA and T3 directly regulate **expression** of these three keratin genes through the action of their **nuclear receptors**. In this paper, we review our previous results to stress that RA has a dual effect on keratin **expression** in epidermis: both direct and indirect. We also analyze the DNA sequences upstream from those three RA-regulated keratin genes and. . . may comprise the putative retinoic acid recognition elements (RAREs). Furthermore, our recent results concerning the regulation of K5 and K14 **expression** by the RA receptor are also shown; these confirm our predictions regarding the location of the RAREs in epidermal keratin. . .

L4 ANSWER 33 OF 33 MEDLINE DUPLICATE 20
ACCESSION NUMBER: 91299797 MEDLINE
DOCUMENT NUMBER: 91299797 PubMed ID: 1712634
TITLE: Nuclear receptors for retinoic acid and thyroid hormone regulate transcription of keratin genes.
AUTHOR: Tomic M; Jiang C K; Epstein H S; Freedberg I M; Samuels H H; Blumenberg M
CORPORATE SOURCE: Department of Dermatology, New York University Medical Center, New York 10016.
CONTRACT NUMBER: AR30682 (NIAMS)
AR39176 (NIAMS)
DK16636 (NIDDK)
+
SOURCE: CELL REGULATION, (1990 Nov) 1 (12) 965-73.
Journal code: A1U; 9005331. ISSN: 1044-2030.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199108
ENTRY DATE: Entered STN: 19910908
Last Updated on STN: 19980206
Entered Medline: 19910820

AB In the epidermis, retinoids regulate the **expression** of keratins, the intermediate filament proteins of epithelial cells. We have **cloned** the 5' regulatory regions of four human epidermal keratin genes, K#5, K#6, K#10, and K#14, and engineered constructs in which these regions drive the **expression** of the CAT **reporter gene**. By co-transfecting the constructs into epithelial cells along with the vectors expressing **nuclear receptors** for retinoic acid (RA) and thyroid hormone, we have demonstrated that the receptors can suppress the promoters of keratin genes.. . . cultures of epithelial cells. The three RA receptors have similar effects on keratin gene transcription. Our data indicate that the **nuclear receptors** for RA and thyroid hormone regulate keratin synthesis by binding to negative recognition elements in the upstream DNA sequences of the keratin genes. RA thus has a twofold effect on epidermal keratin **expression**: qualitatively, it regulates the regulators that effect the switch from basal cell-specific keratins to differentiation-specific ones; and quantitatively, it determines. . .

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